SEVERE LOCALIZED EPIDEMIC OF "INFANTILE PARALYSIS" IN THE TROPICS

BY

W. G. KERR, M.B., F.R.C.S.Ed.
Surgical Specialist, Tanganyika

AND

N. J. PEASE, M.B., B.Chir.
Medical Officer, Tanganyika

Since the last war it has become widely recognized that the low incidence of paralytic poliomyelitis in under-developed countries is associated with an actual hyperendemicity of the disease in those regions. This hyperendemic state is revealed by the sporadic occurrence of cases of "infantile paralysis" and by the immune state of virtually the whole of the native population over the age of 5. The actual number of these sporadic cases is much greater than has often been realized in the past, owing to poor reporting. Quite a number of cases have also been diagnosed as sciatic palsies following the injection of quinine for "fever."

This infantile pattern of infection appears to have been almost world-wide until approximately 1870; since then two distinct changes have been noted, beginning in the more highly developed countries. In the first phase, epidemics of the paralytic disease occurred, still confined to those under 5 and apparently unrelated to the season; in the second phase the age incidence steadily increased, there was a marked increase in the severity of the disease, and it tended to become seasonal. The epidemic described below falls into the first phase of this change—that is, there was a sudden very high incidence of paralytic disease without any alteration in age incidence.

The District

Geographical and Climatic Features.—Nzega Township is the administrative centre of the Nzega District of Tanganyika. It is 4 degrees south of the Equator, but, at 4,000 feet (1,220 metres) above sea-level, there is a subtropical climate all the year round, with a mean temperature of 70° F. (21.1° C.). There are two rainy seasons—a short one normally in November and December, and a longer heavier period from March to May. From May to October very little rain falls. The provincial capital, Tabora, is 75 miles (120 km.) south, and the territorial capital, Dar-es-Salaam, is on the coast 550 miles (885 km.) away. The district itself is some 100 miles (160 km.) long by 60 miles (96 km.) broad (6,000 square miles—15,360 square km.).

Population.—The African population of the district is estimated at 200,000, about half of whom live in the north-west sixth of the district. Indians and Arabs, with a low standard of living, own shops in scattered trading centres throughout the district. The European population consists of approximately 50 missionaries, including their families, in isolated mission stations, a European boarding school at Nzega with 16 pupils, and the Government staff of 20, including families, stationed at the township.

Medical Staff.—The medical facilities are described in some detail to emphasize that conditions in the district are still very primitive. Prior to the epidemic, a health inspector, stationed at Tabora, visited the district about once every three months, and, apart from one African sanitary orderly stationed in the township, no health staff was actually stationed in the district. Little so far has been achieved in the way of providing protected water supplies or improved sanitation, and, while vital statistics are non-

existent, it is believed that the infant mortality rate is in the region of 400–500 per 1,000 births. On the clinical side, "general-practitioner" services are provided by native rural medical aids who work at 13 native authority dispensaries, at four mission stations where there is supervision by European sisters, and at the hospital out-patient department. The 40-bed hospital is in the sole charge of an African medical officer who is responsible for all medical, surgical, and complicated obstetrical cases, and there is also a maternity clinic, under a European health visitor, at which there are some 900 deliveries a year (about 25% of these are abnormal). One of us (N. J. P.) visited the district approximately once every two months prior to the epidemic in a supervisory and advisory capacity.

Onset and Course of Epidemic

The first warning that anything untoward had occurred was the admission to hospital of six cases of paralytic poliomyelitis during the week ending September 25, 1954. Three of these cases came from within a radius of 5 miles (8 km.) of the township and the other three from an area about 17 miles (27 km.) away.

The health inspector and one of us (N. J. P.) proceeded to Nzega to advise on preventive measures and to assess the extent of the outbreak. From the missions, informa-

TABLE I.—Notified Cases of Paralytic Poliomyelitis According to Week of Onset of Paralysis

Week Ending	Week No.	Cases Admitted				Cases Reported from Missions, etc.	Weekly Totals
		Mild	Inter-mediate	Severe	Fatal		
July 31	30					1*	1
Aug. 28	34					2	2
Sept. 4	35			3		3	6
" 11	36		1		1	6	8
" 18	37	1			1	4	6
" 25	38		2				2
Oct. 2	39	2	1	2			5
" 9	40		2	2	3		7
" 16	41	3	2	5	1		11
" 23	42	2	2	4		1	9
" 30	43	1	2	5	1		9
Nov. 6	44	1		1	1		3
" 13	45	2	1	1	2		6
" 20	46		2			1	3
" 27	47	2	2	1		1	6
Dec. 4	48			2	1	1	4
" 11	49	1					1
" 18	50	1					1
" 25	51						Nil
Jan. 1	52		1				1

A 2-months-old case seen in hospital out-patient department.

tion was collected regarding no fewer than 15 other paralytic cases which had occurred during the previous two to three weeks (Table I). The exact date of onset of these cases is unknown, but they have been tabulated, together with the first six admitted cases, as close to the week of onset as could be ascertained.

At the end of September an out-patient was seen with a history of paralysis for two to three months (Table I). This may have been one of the index cases for the epidemic or may have been one of the usual four or five annual sporadic cases.

With intensive propaganda it is believed that the great majority of subsequent cases were admitted to hospital or notified. The epidemic lasted a further 15 weeks, there being 62 admissions and four notifications in the eleven

TABLE II.—Notified Cases in Second Outbreak

Week Ending	Week No.	Cases Admitted		
		Mild	Intermediate	Severe
March 5	9			1
" 19	11	1		
April 9	14		1	
" 16	15		1	
" 23	16		1	
May 7	18			1
" 21	20	1		

weeks to December 4 and three final admissions in the last four weeks of the year. The total notifications in this outbreak were 90, of whom 71 were admitted. All these cases had the paralytic form of the disease.

A second small outbreak occurred in the twelve weeks from February 26 to May 21, 1955, eight cases being admitted, with no deaths (Table II).

In the two outbreaks there were 99 notifications, including the possible index case; 97 of these were Africans, one was an Indian, and one was an Arab. All cases occurred within the north-west sixth of the district, but none within the boundaries of the township itself, although it lay in the centre of the affected area.

Control Measures

While endemic poliomyelitis is most often spread by the faecal-oral route, throat infection and droplet spread probably play an important part in epidemics (McFarlan *et al.*, 1946). The presence of many flies in a cattle-breeding area will increase the risks of spread, and the remote possibility of spread by mosquitoes cannot be ruled out (Paul, 1955a).

Measures were directed to the isolation of the known infected cases, to the improvement of general hygiene, and to efforts to prevent spread outside the affected district. The 68 admitted cases were placed in mosquito- and fly-proof wards, the children were trained to use chamber-pots, and excreta were disposed of in water-borne latrines leading to soakage pits.

In the township the European families were advised to pay particular attention to hygiene, especially in the preparation of food, to keep their children within the small European area, and to keep dogs, which might come in contact with excreta in the bushes, on a leash. The European boarding-school was not closed, but, in the district, all day schools, market-places, and other meeting-places were closed. The digging of pit-latrines was encouraged, and a large number of huts were sprayed with gamma benzene hexachloride to reduce the fly population.

From October 21 to December 20 the infected part of the district was declared an "infected area" under the Public Health Ordinance, and no one was permitted to enter or leave the area. This order could not be rigidly enforced, but was largely effective and may have contributed to a reduction in spread to neighbouring districts. One butcher from Bukoba, 200 miles (320 km.) away, returned to his village from Nzega during November and within four weeks of his return four cases of paralytic poliomyelitis developed amongst infant children of neighbours (Medical Department, 1954). This man may have suffered from an unapparent infection or may have been an uninfected carrier. It is possible to get from Nzega to Bukoba in 48 hours by rail and lake steamer.

Clinical Treatment

The main purpose in admitting the children to hospital was to isolate them, and the majority were discharged as soon as possible after three weeks' isolation. During this time such clinical treatment as was possible was given by the limited staff available. This was principally directed to the saving of life by the prevention, where possible, of respiratory infection, but great attention was also paid to the treatment of malnutrition. As in many tropical countries, children are rarely weaned before they are 18 months old, and they suffer from a marked shortage of proteins from the age of 6 months to 2 years.

A physiotherapist was sent from Dar-es-Salaam for two weeks from November 15. She made muscle charts of the 28 convalescent cases then available and taught the local staff the use of passive exercises, sling exercises, and the construction of plaster-of-Paris leg splints for the control of deformity.

No orthopaedic facilities were then available nearer than Dar-es-Salaam, and it is extremely doubtful whether any form of treatment was carried out after the discharge of the patients.

Follow-up

In July, 1955, both of us visited the district in order to review as many of the patients as possible. Of the 79 patients originally admitted, 9 had died in hospital and 3 were known to have died subsequently. The remaining 67 were sent for, and the fact that 46 (68.7%) reported is a high tribute to the assistance given by the Administration and to the popularity of the nursing care, supervised by the health visitor, at the time of original admission.

The purpose of the visit was to assess the extent of residual paralysis, to decide what treatment, if any, was required, and to arrange for this to be carried out by the general surgeon stationed at Tabora. During the assessment it was constantly borne in mind that a barefooted African should not be encumbered with boots and callipers unless absolutely necessary for locomotion or to prevent severe deformity.

Of the 46 patients, 21 had made a complete recovery and there had been good average improvement in all the others despite the complete lack of treatment for three to nine months. Deformities were slight as yet, and, of course, no noticeable leg-shortening had occurred (the examination of over 100 older cases by one of us (W. G. K.) has led to the belief that this is not a complication to be greatly feared in the Tropics). The 25 patients with residual paralysis or paresis were subdivided as follows: no treatment required, 5; too young for any treatment as yet, 11; tendon transplantation advised, 1; correction of deformity and callipers, 2; callipers only, 6.

An endeavour will be made to trace the 21 patients who failed to report; some of them may have fully recovered, while others may have failed to survive. From a study of the notes it was observed that the cases had been classified as: very severe with associated severe malnutrition, 4; severe, 7; intermediate, 2; mild, 8.

Morbidity and Mortality

Table III gives the age distribution of the 99 notified cases; as birthdays are unknown in many cases, the ages given are not precise but are accurate to within three to

TABLE III.—Age Distribution of Patients

Age	Number	Cumulative Percentage
0-3 months	Nil	—
4-5 "	4	4.0
6-11 "	12	16.2
1 year	36	52.5
2 years	32	84.8
3 "	9	93.9
4 "	2	96.0
5 "	2	98.0
8 "	1	99.0
10 "	1	100.0
Total	99	

four months. There were 48 males, 44 females, and in seven cases the sex was not recorded on the notification form from the missions. It will be seen that 98% of the children were under the age of 6 and 85% under the age of 3.

All the cases occurred within a limited area in the north-west sixth of the district, and this area contains about half of the native population—that is, about 100,000. The incidence of *paralytic* poliomyelitis was therefore 99 per 100,000 population.

The exact proportion of paralytic to non-paralytic infections is unknown, but Melnick and Ledinko (quoted by Paul, 1955b) estimated the ratio as 1:86 between the ages of 1 and 4. If the particular strain of virus were strongly neurotropic the non-paralytic ratio would naturally be lower, but even a figure of 1:50 would mean that 5,000 children under the age of about 5 were infected during this outbreak. From the total absence of cases over the age of 10 it would appear that all adolescents and adults were immune to the causative strain of virus.

Table IV shows the distribution according to clinical severity and the mortality rate among the 79 admitted cases. Mild paralysis is recorded if less than one limb was affected;

TABLE IV.—*Severity of Paralysis, Among 79 Admitted Cases, and Associated Deaths from Poliomyelitis*

Degree of Paralysis	No.	%	Deaths
Mild	18	23	Nil
Intermediate	23	29	Nil
Severe	23	29	1
Very severe	15	19	10
Total	79	100	11

intermediate if there was total paralysis of one limb, with or without paresis in other limbs; severe if two or more limbs were totally paralysed; and very severe if there was extensive limb paralysis together with involvement of respiration.

In addition to the 11 recorded deaths there was one known subsequent death, from other causes, in a child with moderate paralysis. Of the 11 recorded deaths, nine occurred in hospital and two after discharge; both these children were very severely ill with quadriplegia, involvement of respiration, and associated malnutrition. They were taken home from hospital by their parents against advice—a move which was not strongly resisted.

No special treatment was available for respiratory cases, and a further 8 of the 15 patients so affected died in hospital. The only other death was in a 1-year-old child with paralysis of three limbs; she died of bronchopneumonia five days after admission.

The known mortality rate was 11 among 79 admitted paralytic cases (14%) and 10 among 15 with respiratory paralysis (67%).

Discussion

Morbidity

Morbidity is normally recorded as the number of notified cases per 100,000 population and the case-fatality rate as the number of deaths per 100 notified cases. Owing to uncertainty of the true number of non-paralytic cases and the variability in the notification of them, no true comparison can be made between one epidemic and another unless only the paralytic rate is counted. This was strongly emphasized by many speakers at the First International Poliomyelitis Conference in 1948. The same remarks apply to any comparison of the severity of epidemics based on the case-fatality rate.

A further factor militating against accurate comparisons is the geographical limitation of all outbreaks. Thus in 1955 the uncorrected incidence of notified cases was 8.7 per 100,000 for the whole of England and Wales up to the week ending September 17; it was somewhere between 13.6 and 18.6 for the West Riding of Yorkshire, and 97.5 for the County Borough of Barnsley (*British Medical Journal*, 1955). The rates for known paralytic cases are not recorded separately, but they may be estimated, from the weekly summary of infectious diseases, as just over one-half.

Treatment

As noted above, patients were admitted for isolation rather than for treatment, and no special facilities were available for the treatment of respiratory cases. The provision of such facilities for every district is not economically feasible at present, especially in view of the fact that this was Tanganyika's first major outbreak; if, however, an increasing number of outbreaks occur their provision must seriously be considered.

Also, for economic reasons, no treatment at all was given during the late convalescent stage: the review undertaken six months after the first outbreak and three months after the second outbreak showed that little harm was done by this. Approximately 50% had apparently fully recovered, and amongst the remainder few deformities had developed. The need for adequate orthopaedic facilities and constant review after this stage is obvious.

Public Health Control

Considerable doubts have been cast upon the efficacy of any measures of public health control, but it is just possible that the closure of the district may have helped to limit the outbreak geographically. The W.H.O. Expert Committee on Poliomyelitis discussed this point in relation to isolated rural and island communities and stated: "Here not only may measures be exerted against an individual household but it should also be possible to prevent the entry into an apparently healthy community of individuals from infected localities" (quoted by Payne, 1955c).

The small European population in the actual township was fortunate to escape any infection, and the absence of infection amongst Africans living in the town may have been partly due to the fact that, for social reasons, few children would actually be living in the town.

Epidemiology

Contributors to the W.H.O. Monograph *Poliomyelitis* (1955) emphasize the changed behaviour of poliomyelitis dating, approximately, from the Stockholm epidemic of 1887. The steady remorseless spread of epidemics through the world was described by Showman (quoted by Payne, 1955a) as "the incoming tide of poliomyelitis." This invasion has also been accompanied by an upward shift in age incidence. It is now recognized that a major factor in the delay of spread to underdeveloped countries is the presence of hyperendemic infection resulting in a high rate of adult immunity. Gear (1955a) goes so far as to say that "such communities are not liable to epidemics of poliomyelitis."

Improved hygiene in well-developed countries has undoubtedly affected the immune state of the population, and Payne (1955b) also points out that social conditions tend to reduce contacts between children under school age.

The Nzega epidemic, however, was severe, and it occurred in a primitive community where there has as yet been little if any improvement in hygiene. This would appear to give strong support to the views of Sabin (1955a), who believes that we are now facing a disease which has changed its character and which threatens the world by the propagation and spread of more virulent strains than were present in the nineteenth century and earlier. The W.H.O. monograph records many recent epidemics in tropical countries, and in some of these infection seems to have spread from one country to another.

Virology

The statement made by Sabin is worth quoting in full: "It is not at all unlikely that future observations will firmly establish the hypothesis that epidemics of poliomyelitis occur when strains of high virulence are disseminated among populations which are insufficiently immunized by infection with strains that are either avirulent or of low virulence."

Many strains of each of the three main types are known to exist, and Rhodes *et al.* (1955) found 10 out of 100 strains which they tested to be untypable. Variant strains have been produced *in vitro* and *in vivo* by Enders, Koprowski, Sabin, and others, and Sabin (1955b) is now studying naturally occurring avirulent strains of all three types "to determine their relative immunogenic and mutagenic properties."

The Nzega outbreak appears to have been caused by a virulent strain antigenically similar to the relatively avirulent strain or strains which had immunized the great majority over the age of 5. Its source is unknown, but it may have been introduced from outside, it may have resulted from the activation of a dormant strain, or it may have been due to a natural mutation from the strains already present.

Introduction from outside is supported by the knowledge of the centrifugal spread of the more virulent disease and by the example of recent West African epidemics (Gear, 1955b) and the 1942 epidemic in Malta (Gear, 1955c;

Sabin, 1955c). One of us (W. G. K.) also observed that the small epidemic in Kenya in December, 1947, may have acted as a link between the major epidemic in Britain in the autumn of 1947 and that in South Africa in the spring of 1948.

Activation of a dormant strain is rendered possible by the known presence of virulent strains in underdeveloped countries. These give rise to sporadic cases of infantile paralysis and caused a high incidence among troops posted to such areas during the last war.

Mutation in culture is infrequent but does occur (Sabin, 1955d), and has almost certainly occurred in nature during the past 60 years. It is unlikely, however, that a local mutation has been responsible for each one of the very large number of epidemics that have occurred during this period.

Future Outlook

The probability of further spread of virulent strains with an increase in the number and severity of epidemics can only be described as grave, and will place a great strain on the resources of undeveloped countries in the provision of treatment both for the acute cases and for an increasing number of cripples.

It would appear that this spread will continue unchecked until there has been produced a vaccine which can provide immunity against all known types and which is within the economic means of underdeveloped countries. Work at present being carried out by Koprowski, Sabin, Dick, and others on the production of an oral vaccine of avirulent strains holds out more hope than do the vaccines in current use owing to the possibility of achieving lifelong immunity by the administration of only one dose (Sabin, 1955b). Further economies in administration and in the cost of a campaign would be achieved if a simple test could be devised for the presence of previously developed immunity in individuals.

Summary

A description is given of an acute localized outbreak of poliomyelitis in a primitive community. The incidence was 99 per 100,000 population, referring to paralytic cases only; 85% of these were under the age of 3 and 96% under 5.

The conclusion is reached that the epidemic resulted from the appearance of a strain more highly virulent than that normally present, although antigenically similar. The occurrence was not due to any marked improvement in social or hygienic conditions.

An increased demand for curative and orthopaedic services is envisaged in underdeveloped countries. The need for orthopaedic services to care for cripples from previous epidemics will continue for many years after an effective and lifelong vaccine becomes freely available.

We wish to thank the Director of Medical Services, Tanganyika, for permission to publish this paper.

REFERENCES

- British Medical Journal*, 1955, 2, 857.
 Gear, J. H. S. (1955a). In *Poliomyelitis*, p. 34. W.H.O. Monograph No. 26. Geneva.
 — (1955b). *Ibid.*, p. 35.
 — (1955c). *Ibid.*, p. 48.
 McFarlan, A. M., Dick, G. W. A., and Seddon, H. J. (1946). *Quart. J. Med.*, 15, 183.
 Medical Department, Tanganyika (1954). Annual Report. Government Printer, Dar-es-Salaam.
 Paul, J. R. (1955a). In *Poliomyelitis*, p. 20. W.H.O. Monograph No. 26. Geneva.
 — (1955b). *Ibid.*, p. 15.
 Payne, A. M.-M. (1955a). *Ibid.*, p. 374.
 — (1955b). *Ibid.*, p. 377.
 — (1955c). *Ibid.*, p. 387.
Poliomyelitis (1955). W.H.O. Monograph No. 26. Geneva.
 Rhodes, A. J., Wood, W., and Duncan, D. (1955). *Ibid.*, p. 246.
 Sabin, A. B. (1955a). *Ibid.*, p. 314.
 — (1955b). *British Medical Journal*, 2, 160.
 — (1955c). In *Poliomyelitis*, p. 313. W.H.O. Monograph No. 26. Geneva.
 — (1955d). *Ibid.*, p. 327.

ETHYLENE GLYCOL POISONING WITH MENINGOENCEPHALITIS AND ANURIA

BY

IAN P. ROSS, M.D., M.R.C.P.

Late Captain, R.A.M.C.; Medical Registrar, the London Hospital

The purpose of this paper is to record and comment on the features of ethylene glycol poisoning as seen in a patient exhibiting meningoencephalitis and anuria, resulting in death after 12 days. In most cases of poisoning death ensues rapidly, and only a small minority survive the initial phase and die later from renal failure.

Ethylene glycol is a clear, colourless, odourless liquid with a bitter-sweet taste, and produces a sense of warmth when drunk. It has the formula $\text{HOCH}_2\text{-CH}_2\text{OH}$, a boiling-point of 197°C ., is heavier than water, and evolves heat when mixed with it. Ethylene glycol is one particular substance from a whole series of glycols, and chemically it occupies a position between ethyl alcohol and glycerol. Its main uses are as an antifreeze agent, as a solvent, and in electrolytic condensers. The properties, uses, and animal toxicity have been fully reported by Hanzlik *et al.* (1931) and Browning (1952).

Human poisoning with it has been known for about 30 years. Most cases have occurred as a result of drinking "antifreeze," and this has been especially common among Service personnel; it has been taken by accident, but has often been drunk as a substitute for alcohol. Series of fatal cases have been reported by Boemke (1943), Pons and Custer (1946), Kahn and Brothner (1950), Smith (1951), and others.

Case Report

At 4 p.m. on February 4, 1953, a healthy man of 33 was seen to drink $\frac{1}{4}$ – $\frac{1}{2}$ pint (140–280 ml.) of antifreeze solution containing almost pure ethylene glycol. Five hours later he appeared ill and became drowsy; he was put to bed and slept peacefully. The next day he was very drowsy, and by noon was in a coma; the temperature was 95°F . (35°C .), pulse rate 80, respiratory rate 20, and blood pressure 110/70. At this time the reflexes were brisk and equal and the plantar responses flexor. Eight hours later he was less drowsy, but was delirious, and reacted to painful stimuli but not to commands. Bruises were noted on the arms and legs. The temperature had risen to 102°F . (38.9°C .) and the blood pressure to 160/80, the pulse and respiratory rates being almost unchanged. There was some twitching of the face and arms, and moderate neck rigidity. The pupils were small and equal in size, and responded sluggishly to light. The remainder of the nervous system was normal, but there was incontinence of urine. A lumbar puncture showed a slightly turbid cerebrospinal fluid (C.S.F.) under increased pressure. Analysis showed 400 mg. of protein and 30 mg. of glucose per 100 ml. The total cell count was 695 cells per c.mm. (80% polymorphs, 20% lymphocytes). A blood count showed a haemoglobin of 96% and a total white-cell count of 28,000 (85% polymorphs).

On February 6 he was restless but less drowsy, and his temperature was 99°F . (37.2°C .). He had taken fluids by mouth, but had vomited a little. There was less neck rigidity, but the twitching persisted in the face and also in the arms, which were now adducted and flexed. The limb reflexes were very brisk, the abdominal reflexes absent, and the plantar responses extensor; there was bilateral knee and ankle clonus. The liver was felt one fingerbreadth below the costal margin. As he had passed no more urine, he was catheterized and 150 ml. of turbid amber urine of S.G. 1012 obtained. This contained one-eighth volume of albumin but was otherwise normal; there were a few leucocytes in